ScienceMind

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KING'S College LONDON

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THIS ISSUE

Dear Reader,

Welcome to the third issue of ScienceMind (previously KCL Pharmacologist). A big thanks to the writing, editing and media teams for helping to contribute to this issue.

If this is your first time reading our magazine...
Science Mind is the award-nominated, student-led science magazine of King's College London, which focuses on reporting recent findings in the main branches of science to students and the wider community. We aim to showcase and develop the written and oral communication skills of students interested in research by concisely explaining complex scientific concepts in the form of lay articles and conducting interviews. Authors can also broaden their knowledge by writing articles for different sectors between issues.

If science communication is a concept that interests you, I encourage you to join the dynamic and constantly expanding team of ScienceMind. We would love to have you on our team!

Yours faithfully,

The Editor-in-Chief
The Founder

CONTENTS

DEEP DIVE

COULD GRAPEFRUIT JUICE BOOST THE BLOOD PRESSURE LOWERING EFFECTS OF BEETROOT JUICE?

WRITTEN BY
RACHEL BRADY

Dietary nitrate, sourced from beetroot juice and other green leafy vegetables like spinach, have been shown to lower blood pressure via its reduction to nitrite and nitric oxide (NO) in the nitrate-nitrite-NO pathway. However, re-oxidation of nitrite (NO2-) and NO to nitrate (NO3-), negates their blood pressure lowering effects. Cytochrome P450 enzymes are heavily involved in drug metabolism. The CYP3A4 isoform found in the gut enterocytes and liver, is thought to be involved in nitrite oxidation.

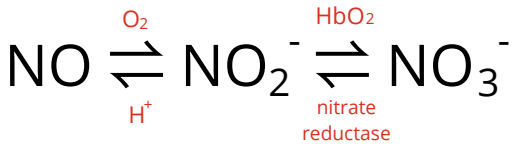

Figure 1. The nitrate-nitrite-NO pathway

A previous study in rat liver homogenates by Curtis et al. in 2012, showed that inhibiting CYP3A4 led to the inhibition of nitrite re-oxidation to nitrate with agents such as troleandomycin (a cytochrome P450-3A4 inhibitor). Similarly, grapefruit juice is also known to interact with drugs by inhibiting CYP3A4 due to furanocoumarins in the juice. Therefore, it was hypothesised by O'Gallagher et al. (2020) from King's College London that co-ingesting grapefruit juice with beetroot juice would, in theory, decrease oxidation of nitrite to nitrate caused by CYP3A4 and cause an increased plasma nitrite concentration (and a potentiated decrease in blood pressure). Beetroot juice itself contains nitrate which is metabolised into nitrite (in the mouth) and NO (in the stomach) which has been shown to lower blood pressure.

The study design chosen was a 3-visit randomised, single-blind, placebo controlled crossover intervention. Patient visits lasted 7 hours in which blood pressure readings, plasma and saliva samples were collected over the course of this timeframe. The design of this study was such that the subjects had to attend all 3 visits; though only 9 out of the 11 patients attended all 3. At each visit. the patients consumed one of either:

- Nitrate-containing beetroot juice + Grapefruit juice
- Nitrate-containing beetroot juice + Water
- Nitrate-depleted beetroot juice + Grapefruit juice

ABOUT THE AUTHOR

Rachel Brady is a 2nd year BSc Pharmacology student with an interest in cardiovascular pharmacology and immunopharmacology.

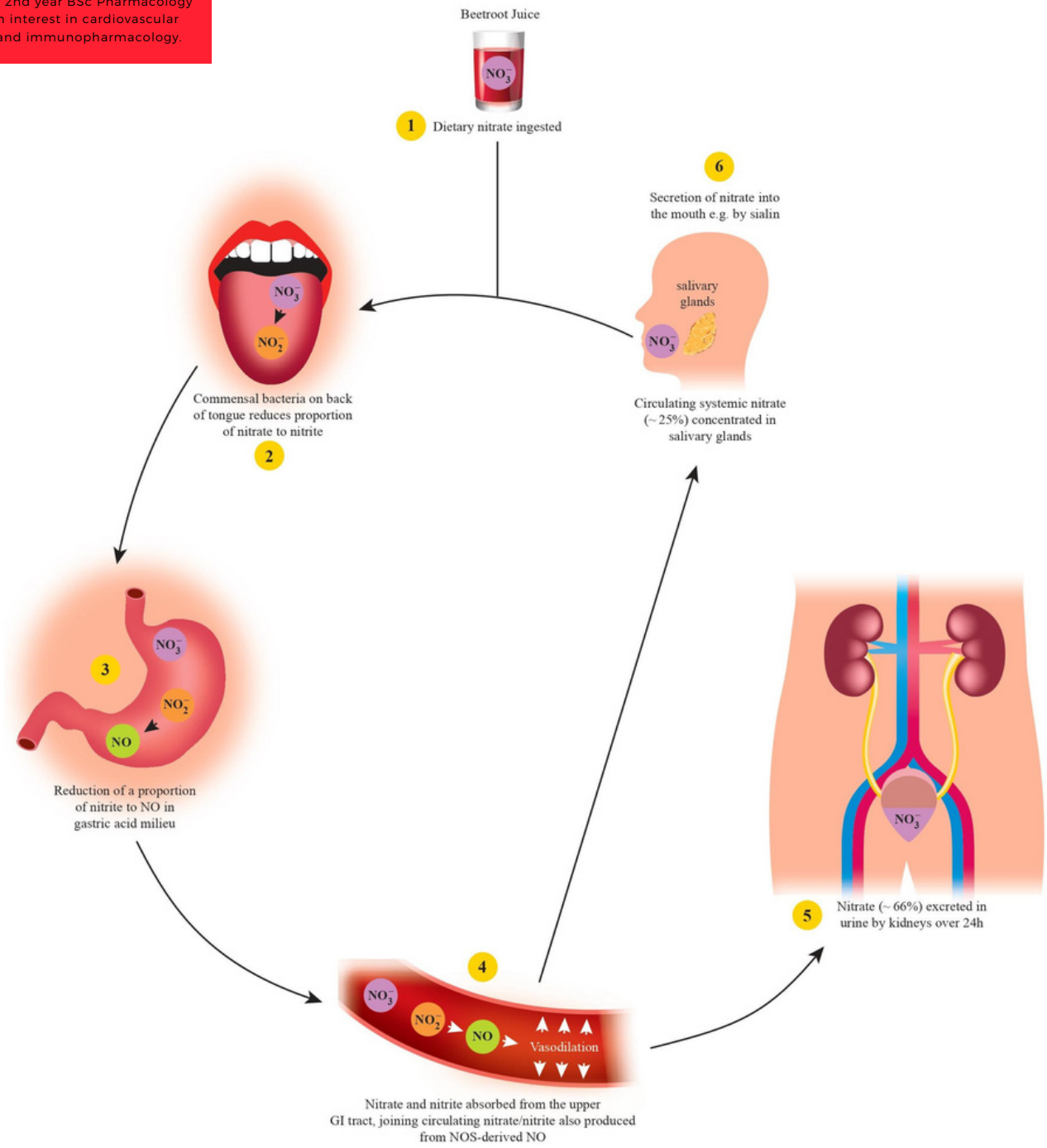

Figure 2. Stages of the enterosalivary circulation and how it ties in with the nitrate-nitrite-NO pathway *(Chirinos, Textbook of Arterial Stiffness and Pulsatile Haemo-dynamics in Health and Disease, 2022, Elsevier).*

O'Gallagher and colleagues found that **grapefruit juice potentiated** the systolic blood pressure lowering effect of beetroot juice but strangely showed a **decrease** in plasma nitrite concentration. The findings are interesting as in theory, it would be expected that this decrease in blood pressure would be caused by the **increase** in plasma nitrite concentration after **inhibiting CYP3A4**.

In addition, they found that grapefruit juice actually **inhibited** the metabolic reduction of nitrate into nitrite so the blood pressure lowering effect could be due to other NO species that were not investigated in this study. In sum, these findings suggest more complex mechanisms behind dietary nitrate bioactivation but equally show that dietary nitrate has **potential** clinical benefit for use in **targeting systolic hypertension**.

THREADING WATER

THE BRAND NEW DRUG FOR NARCOLEPSY: SOLRIAMFETOL

WRITTEN BY AAIMAN BHARMAL & SEAN CRAWFORD

Credits: American Pharmacy News (2020).

Narcolepsy is primarily characterised by excessive sleepiness (ES) as well as cataplexy (brief loss of muscle tone triggered by strong emotions). Current medications for the treatment of narcolepsy are used to relieve these symptoms and therefore significantly improve the quality of life of narcolepsy sufferers. The American Academy of sleep medicine has termed ES management in narcolepsy of utmost importance.

Solriamfetol, a selective dopamine and noradrenaline reuptake inhibitor has been recently approved to treat ES.

From preclinical and clinical trials it has been suggested that Solriamfetol promotes wakefulness in a manner which is different from current reuptake inhibitors such as modafinil and cocaine, alongside other stimulants such as adderall (amphetamine salts), which are all used to treat ES.

The underlying neurobiology

The epidemiological occurrence of narcolepsy in North American, Western European, and Asian populations is 0.2% to 0.67%.

Structural imaging studies on the brains of patients with narcolepsy have revealed localised decrement of grey matter in the hypothalamus, nucleus accumbens, frontotemporal cortices and thalamus. The hypothalamus contains hypocretinergic neurons which are key excitatory components of neuronal circuits that control sleep-wakefulness cycles. These neurons produce the Hcrt/Orx neuropeptide, which in low abundance/ absence has been linked to the major symptoms of narcolepsy. The rest of the brain areas affected are key sites innervated by the hypocretinergic neurons. The hypocretinergic neurons in these areas have excitatory effects on serotonergic, noradrenergic, histaminergic and cholinergic neurons which are all part of the wakefulness promoting circuit, and additionally, they exhibit inhibitory effects on REM sleep generation.

By the result of many environmental influences and genetic factors, it has been implemented that malfunctioning sleep-wake cycle control is induced by an immunogenic reaction against the hypocretinergic neurons, which ultimately ceases the inhibitory effects on REM sleep generation hence prompting excessive sleepiness.

Solriamfetol's effects on inducing wakefulness have been studied through its antagonist effects on the uptake 1 receptors for DAT, NET and SERT.

These receptors are responsible for the transport of neurotransmitters back into the presynaptic nerve terminals where they were initially released from. Blocking this uptake increases neurotransmitter concentration in the synapses which helps to activate the systems involved in wakefulness, preventing the patient from falling asleep.

The preclinical assays

As part of preclinical studies outlining the binding specificities and the efficacy of Solraimfetol, a number of assays were carried out. Binding of Solriamfetol was assessed by in vitro transporter assays using human embryonic kidney cells expressing DAT, NET and SERT.

Furthermore, monoamine reuptake inhibition and release assays revealed some interesting insights too. These demonstrated Solriamfetol's receptor interactions involving dopamine and noradrenaline. These suggested that Solriamfetol was a less potent inhibitor than cocaine which was a positive result in terms of Solriamfetol's use for clinical applications of treating ES. Additionally through the release assays on preloaded transfected cells it was confirmed that the medicine had no effect on release of dopamine, noradrenaline, and serotonin hence indicating specific action of drug to solely inhibit reuptake.

In mouse behavioural studies involving cocaine, saline and then cocaine being substituted with Solriamfetol, suggested evidence that Solriamfetol is less addictive than cocaine. This evidence is backed by in-vivo transporter and receptor assays. These results showed that Solriamfetol's binding affinity for DAT and NET was 60 fold and 7 fold respectively lower than that of cocaine. Which means that the drug did not stay bound to the receptor for very long time implying it will not lead to build up of dopamine levels to the extent of causing euphoria. Also solriamfetol had negligible binding affinity for SERT receptors. This absence of serotonergic mechanism of action suggested that Solriamfetol induced effects were different from those of fluoxetine which is used as a stimulant to treat ES.

Clinical Trials (Results and adverse effects)

During phase 2 and 3 clinical trials a total of 226 patients with narcolepsy and 417 patients with obstructive sleep apnoea were subjected to long term 52 week treatments with Slraimfetol. These showed a continuous reduction in scores calculated on the Epworth Sleepiness Scale compared to the placebo group. Post treatment at 6 months: 280 patients entered a 2 week placebo controlled withdrawal period which led to an increase in the sleepiness scores to 5.3 which can be compared to a - score of 1.6 obtained from those patients which were kept on active treatment with Solriamfetol.

During the trials adverse reactions to the medication were headache, nausea, anxiety, insomnia, and decreased appetite. Also the trials revealed pharmacodynamic properties of the drug involving excretion and metabolism. The medicine has been shown to be minimally metabolised in humans to N-acetyl Solriamfetol and 95% of the medicine is excreted unchanged by the kidneys with the clearance rate being 18.2 L/hour.

Solriamfetol now marketed as Sunosi, which was discovered and developed by SK biopharmaceuticals in South Korea. The current holder of rights for Solriamfetol is Jazz Pharmaceuticals who got approval from the FDA and European Commission to market the drug as a sleep medication for excessive sleepiness occurring in narcolepsy and obstructive sleep apnoea earlier in 2020.

ABOUT THE AUTHOR

Aaiman Bharmal is a 1st year BSc Pharmacology student interested in drug discovery and development.

Sean Crawford is a 3rd year BSc Pharmacology student with interests in neuropharmacology.

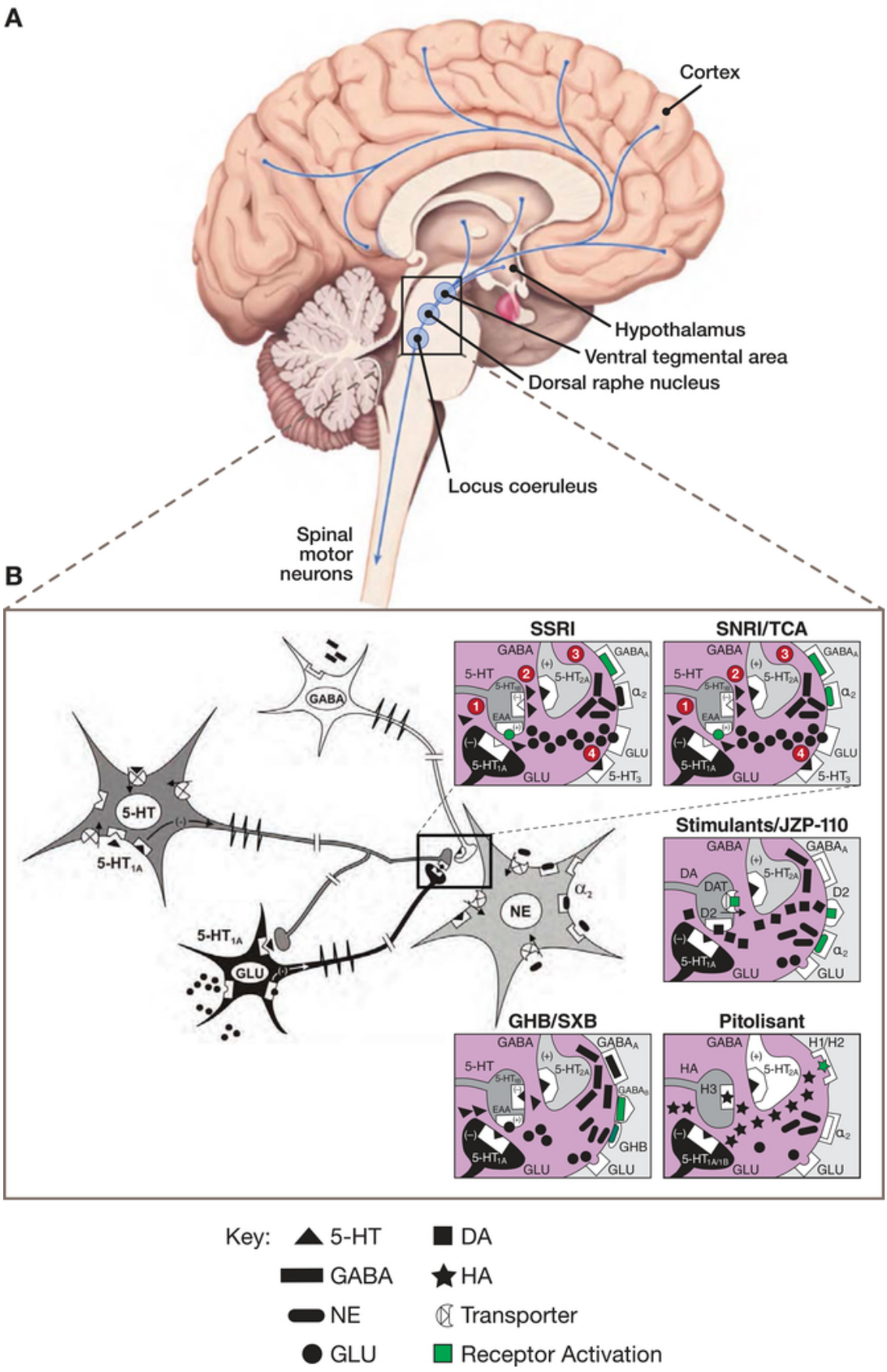

Figure 1. A) diagram depicting which nerves innervate the brain and B) showing the mechanism of action of these drugs.

"Stay true to yourself and never doubt your principles. Exercise your judgement and you'll get where you want to be!"

DR MICHAEL CURTIS

ISSUE 3 | 2020

Dr Michael Curtis is a Reader in Pharmacology within the School of Cardiovascular Medicine & Sciences, Faculty of Life Sciences & Medicine. He became a lecturer in Pharmacology at King's College London in 1989, and reader in 1996.

His main areas of research include antiarrhythmic and proarrhythmic drugs as well as ischaemic heart disease and Torsades de Pointes. He has published over 100 papers, which have been cited over 5000 times.

THREADING WATER

Cardiovascular Safety Pharmacology Paradigms: Is the New the Well Forgotten Old?

DR MICHAEL CURTIS TALKS TORSADES DE POINTES AND DRUG DEVELOPMENT

WITH PETR BORODAVKIN

The estimated expenses needed to develop a new drug and successfully bring it to the market have been suggested to cost pharmaceutical companies a whopping $1.3 billion. Given such enormous stakes, the process of drug development itself has undergone serious transformations to include additional safety investigations to identify and, if possible, neutralize the dangerous effects of potential drug candidates as early as possible.

One of such methods is the hERG assay- a procedure commonly performed during pre-clinical development to determine the test compounds' potential for triggering undesired cardiovascular complications. hERG particularly investigates the effects on the cardiac rapid delayed rectifier channel known as IKr. Inhibition of IKr has been linked to Torsades de Pointes – a cardiac arrhythmia leading to sudden death, which will in turn nullify the chances of the drug ever reaching human patients if it is discovered as one of its side effects.

We sat down with Dr. Michael Curtis from the Cardiac Pharmacology Department here at King's College London to discuss the impact of Torsdades de Pointes on the development of safety pharmacology and drug development industry.

"The idea of the drug producing a lethal adverse effect is always a horror scenario for the [drug] development team regardless of what it is." reflects Dr. Curtis.

This was certainly the case when Torsades de Pointes was documented for the first time in people taking terfenadine- an antihistamine drug with off target inhibitory effects on the IKr channels, leading to fatal arrhythmias in 50 unsuspecting patients in the early 1990s, which forced the drug to be immediately removed from the market. This tragedy has since become the reason why all drugs going through development are screened for IKr inhibition through hERG.

Although allowing to screen out potentially hazardous drugs at an early stage and minimize financial losses, hERG has come under scrutiny as pharmacologists expressed their concerns that the procedure may have allowed many potentially safe drugs to be wrongfully discarded from further development, calling for the assay to be modified. A commonly used example in favour of this theory is verapamil- an extremely popular anti-hypertensive drug with no recorded torsadogenic effects, amassing 4 million prescriptions in the US alone in 2017. However, verapamil has been found to have inhibitory effects on IKr, suggesting that it would never reach the market if it was put through hERG during its development. Therefore, a question arises whether it is possible that other non-torsadogenic drugs were falsely discarded since the introduction of hERG assays into the drug development pipeline in the late 1990's.

Dr. Curtis finds this schism to be rooted in the policies of drug companies rather than available scientific evidence on the topic: "It's simply based on the existence of verapamil. If you're going to use verapamil as your exemplar in developing better diagnostic assays, all you need to do is a hERG screen, and then screen for effects on the L-type calcium channel if you get an inhibitory effect on IKr. If the drug mimics verapamil, which is known to be safe, there shouldn't be a concern" he says.

"What happened instead, is that the drug companies received funding and committees were established to screen for every channel under the sun, making more problems for themselves, rather than solving the existing ones".

In addition, the chemical structures known to be associated with Torsades de Pointes could be manipulated by medical chemists in order to avoid its manifestation when taken by humans, supporting the claim of Dr Curtis that even if verapamil was rejected based on the results hERG assay, its chemical structure would be altered so that it could no longer inhibit IKr, while being perfectly capable of exerting it's effects, instead of discarding the drug.

However, the way the issue is being approached by pharmaceutical companies may be seen as at least thought-provoking. Dr Curtis' take on the situation enhances this notion: "It's a very interesting thing to look at. Drugs are being developed and safety-tested not on the basis of sound scientific principles but on the basis of how the safety testing fits into the marketing model the company is operating". Coming back to the hERG dilemma, the new modifications would in this context allow companies to keep their funding, despite there being no potential need for it.

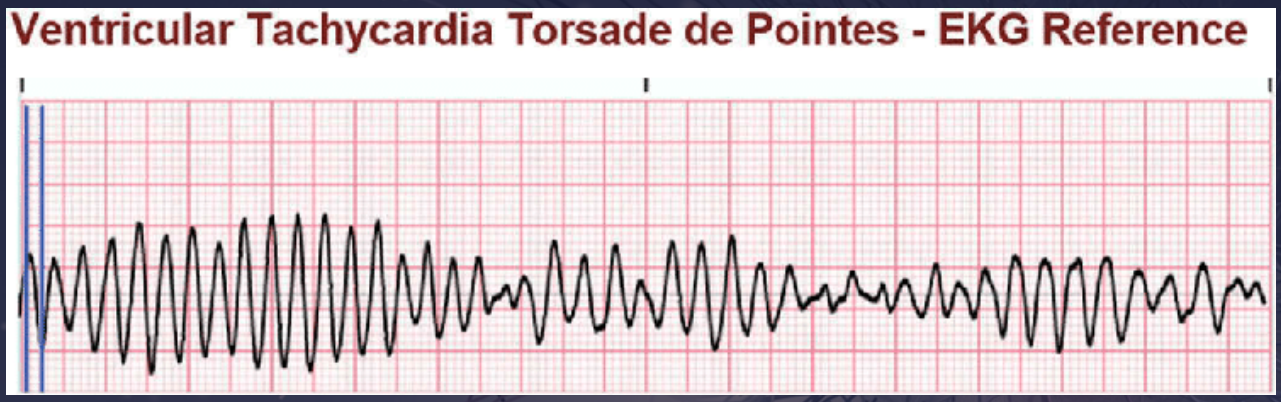

Figure 1. EKG from a patient with ventricular torsades de pointes.

"It would certainly be useful if drug companies explained the logic and reasoning behind how they chose different models and approaches of how to develop a drug but what I've found is that they can't or don't publish that information" he adds, when asked how the companies may address the issue and avoid procedures which might not be necessary to ensure greater safety of the drugs produced.

Nevertheless, Dr Curtis remains optimistic about the future prospects of drug discovery and suggests that although there may be problems within the industry, they will not affect the impact of research in academia: "In order to understand the present you always need to look in the past. Drug discoveries have been generally made through the endeavours of small teams of people trying to do things properly and/or they [also] have been lucky. It never really worked in the corporate way".

In fact, the following framework perfectly reflect the research of Dr Curtis, as he shared with us of one of his current projects.

The topic at play is a lidocaine-based pro drug and its potential in the management of ischaemia, which has long been an area of expertise for him. The exciting part, however, is that the drug will remain inert when administered to patients and only get activated by the onset of ischemia. This could meet an unmet clinical need, as one of the main challenges of ischemia management is rapid treatment after its onset. "The ischemia traps the drug and activates it, allowing it to prevent arrhythmias, without displaying any side effects of lidocaine" happily reflects Dr Curtis. After all, he admits that the most important piece of advice is to stay true to yourself and never doubt your principles: "Exercise your judgement and you'll get where you want to be!" which undoubtedly brings reassurance to the upcoming pharmacologists and will hopefully captivate them to strive to get to the top of their future areas of expertise.

ABOUT THE AUTHOR

Petr Borodavkin is a 3rd year BSc Biomedical science student familiar with T2DM and BPD.

THREADING WATER

WHY DO WE NEED SUNLIGHT TO STAY HEALTHY?

WRITTEN BY SOUMIYA DRIR SADAOUI

Vitamin D (the sun vitamin) first appeared 2.1 billion years ago after the 'Great Oxidation Event' when the first eukaryotic bodies originated; the rise in atmospheric oxygen subsequently enabled the synthesis of cholesterol. Vitamin D is the result of exposing 7-dehydrocholesterol (the cholesterol precursor) to the sun. Hence, vitamin D is a steroid hormone made up from cholesterol (figure 1).

Vitamin D sources can be found in our diet; D2 comes from plants and D3 from animal products or it can be synthesised by our cell skins when exposed to the sun. In the liver specific enzymes convert vitamin D3 into 25(OH)D3 by adding an OH group to the position number 25 of the chemical structure shown in figure 2. 25-hydroxycholecalciferol or 25(OH)D3 is the most stable form of vitamin D in our body, and thus the most abundant form in our blood.

Maintaining an adequate level of vitamin D in our body is crucial to protect our immune system, as well as the state of our bones. This steroid hormone regulates antimicrobial properties and modulates the action of adaptive immunity, which is the type of immunity our bodies acquire after being exposed to an antigen (for example after vaccination). Adaptive immunity is necessary to prevent autoimmune disease where the body mistakenly attacks your own body, causing an illness.

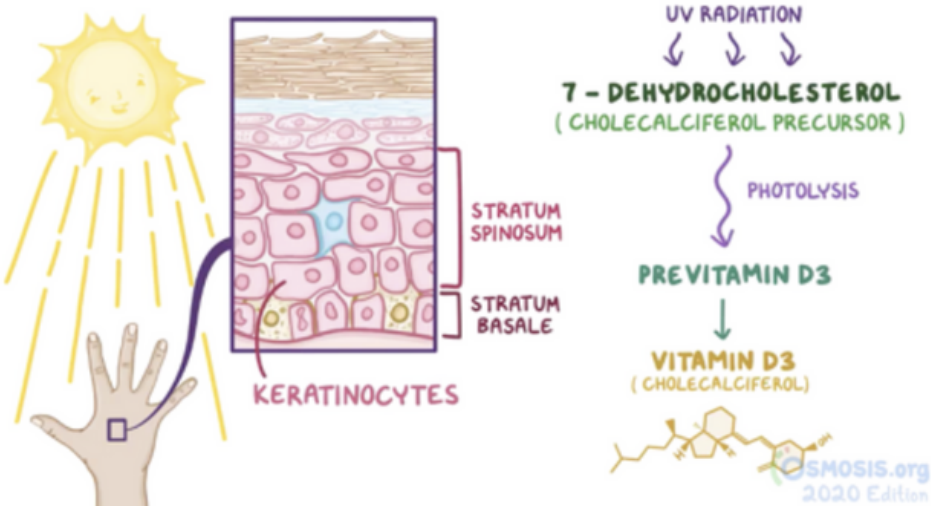

Figure 1. Illustration of the process by which Vitamin D3 is synthesised (Adapted from Osmosis 2020).

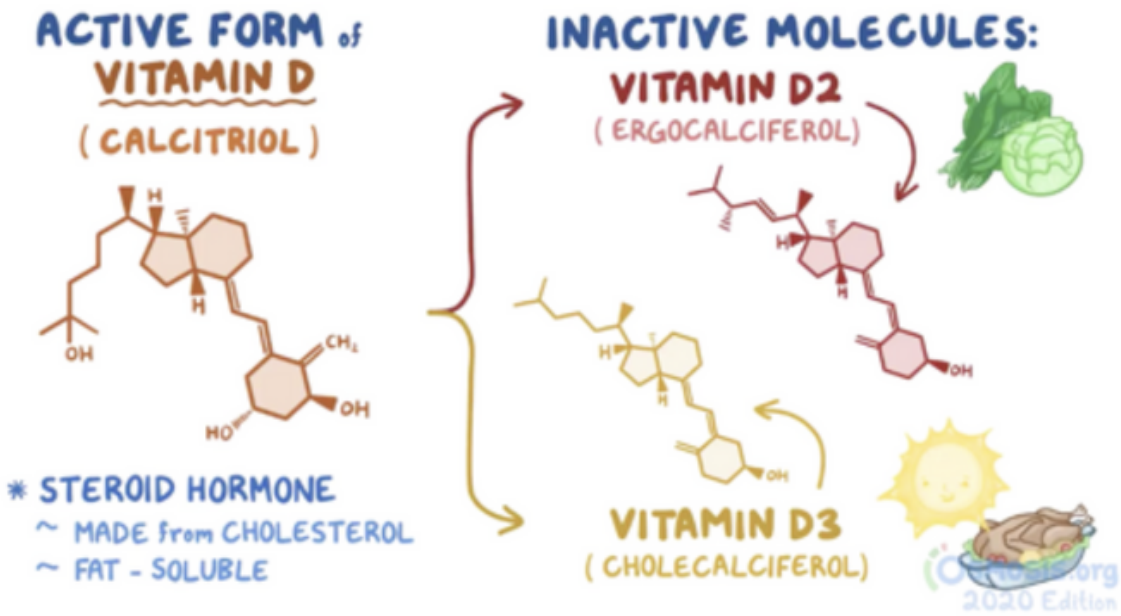

Figure 2. Illustration of the active and inactive forms of Vitamin D (Adapated from Osmosis 2020).

A few examples of these autoimmune diseases include rheumatoid arthritis and coeliac disease. In addition, vitamin D also regulates the reabsorption of osteoclasts which are bone macrophages (i.e.. white blood cells which destroy harmful organisms). Behind the serum levels of vitamin D there is a long evolutionary story which started as a photochemical reaction due to oxygen. Over the years, it gained an endocrine role and an immune-regulatory one which is now what contributes to a healthy state of our bones. It is important to note that different races require different amounts of vitamin D. In fact, lower sun exposure in high latitudes favour reduced skin pigmentation and permits sufficient vitamin D to be synthesised in the body.

However, it does not favour dark skin pigmentations who would only synthesise normalised range levels of vitamin D at the equator. These days, our new adopted lifestyles, being mainly indoors and reduced meat in our diets requires us to take vitamin D supplements to support our body.

ABOUT THE AUTHOR

Soumiya Drir Sadaoui is a 2nd year BSc Pharmacology student. Her main focus is on mental health and neuropharmacology in general..

THREADING WATER

THE SELECTION OF A CANDIDATE BETWEEN TWO RNA-BASED COVID-19 VACCINES

WRITTEN BY CHARLINE HENDRICKX & CHETANA PRABHU

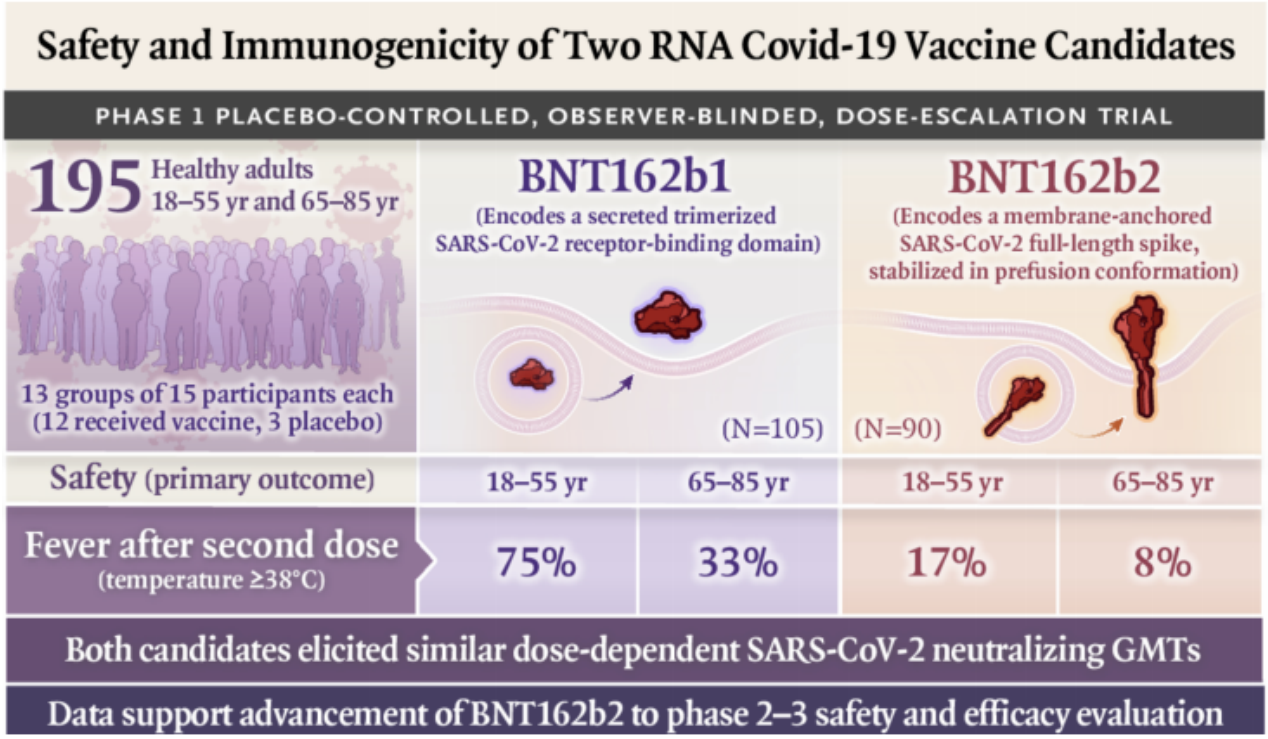

Figure 1. "Safety and Immunogenicity of Two RNA Covid-19 Vaccine Candidates"

The world desperately needs a Covid-19 vaccine and one is well on its way. A program was designed by BioNTech and Pfizer to select a vaccine candidate along with a dose level, to test in safety and efficacy trials. Reported data of a lipid nanoparticle-formulated, nucleoside-modified RNA (modRNA) candidate termed BNT162b1 showed promising value as a Covid-19 vaccine. Two modRNA, BNT162b1 and BNT162b2, are now being evaluated for their safety and immunogenicity in Phase 1 trials in order to select one candidate for Phase 2 trails.

The Phase 1 trials were carried out as a randomised, placebo-controlled, observer-blinded, dose-escalation trial between healthy adults 18 to 55 years of age and elderly of ages 65 to 85. The participants were randomly assigned to groups and received two doses of 10, 20, and 30 µg of BNT162b1, BNT162b2, or placebo 21 days apart. 13 groups with 15 participants were generated with a total of 195 participants taking part in the study. In each group, 12 individuals received two doses of the vaccine and 3 received two doses of placebo.

Safety was assessed observing local reactions and systemic events. BNT162b1 elicited similar local reactions between the participants in both age ranges. The local reactions consisted of mild to moderate pain at the injection site occurring within 7 days of the injection, and more frequent with the administration of the second dose. Similar patterns were observed with BNT162b2. The systemic events seen in the participants 18 to 55 years of age with BNT162b1 included fever and chills with 75% of the participants reporting a fever (38˚C). The systemic events in volunteers aged 65 to 85 years old were milder, and included fatigue and headaches with only 33% reporting a fever. In contrast to BNT162b1, only 17% of the participants 18 to 55 years old, and 8% of the elderly participants reported a fever when administered with vaccine BNT162b2. Needless to say, more severe systemic events such as headaches, muscle pain, and fatigue were seen with BNT162b2 on younger volunteers, however at very low numbers.

The immunogenicity responses were observed and both candidates demonstrated similar serologic responses (changes in blood serum proteins/antibodies). The virus-neutralising response seen with all doses of the vaccines were increased and optimised with the second administration in both participant age groups. It was also seen that a higher dose seemed to generate a higher antibody response.

BNT162b2 displayed a favourable balance between the immune response and the toxicity observed and therefore was chosen to take forward to Phase 2 and 3 trials at the highest dose level tested of 30 µg.

The main factor that led to this decision were the milder systemic reactions observed with BNT162b2 especially in the older participant group. There were some limitations within this study but these have been taken into account moving forward to Phase 2.

ABOUT THE AUTHOR

Charline Hendrickx is a 3rd year BSc Pharmacology student with an interest in oncology and depression in neuropharmacology.

Chetana Prabhu is a 1st year BSc Pharmacology student. Her current areas of interest include neuroscience, genetics and pharmacology.

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KCL PHARMACOLOGY SOCIETY

Happy Holidays!

Hurray for getting through the semester! It's time to enjoy a little break. We can't wait to see you again next semester, so here's a sneak peak to our future events :)

Speed Meeting with Oxford University and St George's University Pharmacology Societies

Negotiation in the Pharmaceutical Industry
Collab with KCL Negotiation Society

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